ALCOHOLISM

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ORATION LECTURE

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ALCOHOLISM

GENERAL INTRODUCTION:

Alcohol is a central nervous system depressant and intoxicant, is the most commonly used psychoactive substance in both the mentally healthy and mentally ill persons. More than 2/3 of the people consume alcohol and the distinction between recreational or social drinking and alcohol abuse is often vague and unclear.

Alcohol is the exogenous toxin most commonly encountered in psychiatric practice, it causes a wide range of nervous system disorders.

An estimated 10 million persons in U.S.A. suffer from alcoholism, alcoholism is clearly the most serious of all drug problems in our country in terms of loss of productivity, the total human tragedy, the extent of physical damage and the No.of fatalities from withdrawal symptoms. The statistics in U.S.A. are awesome: 50% of all fatal automobile accidents, 65% of murders, 35% of rapes, 30% of suicides, 40% of assaults and 60% of cases of child abuse involve alcohol. An estimated 3.3 million teenages in U.S.A. are showing signs of early alcoholism. our country closely correlates with the above statistics of U.S.A.

The name "Alcoholism" was introduced by Magnus Huss, a swedish Public Health Authority in 1849, the word was quickly adopted by many languages.

To understand alcoholism, it is useful to know about alcohol itself and its fate in the body. When Yeast is grown in Sugar solutions without air, most of the sugar is converted (fermented) into CO2 and alcohol. When alcohol concentration reaches about 12 or 13%, the process stops. For this reason, unfortified wines have alcohol concentration of no more than 12 or 13%.

As a rule, people do not drink only alcohol, they drink alcohol beverages, alcohol beverages are mainly water and ethyl alcohol. Tiny amounts of other chemicals are present, providing most of the taste and all of the colour, if any, called cogneres. These chemicals include aminoacids, minerals, vitamins and methanol.

Beverages differ according to the sugar source. Wine comes from grapes, Beer from grain and hops, Whisky from grain and corn, Rum from sugar cane and Vodka from potatoes and grain.

Distillation was discovered about 800 A.D. in Arabia. Alcohol comes from Arabic word "ALKUHL" meaning essence. Distillation boils away alcohol from its sugar bath and recollects as virtually pure alcohol. The water is then put back into the solution, so that instead it being 100% alcohol, the solution is perhaps 50% alcohol or 100% proof alcohol.

One unit of alcohol is 10 ml. which is 8 gms. A unit of alcohol is one single whisky, one glass of Sherry or fortified wine, one glass of wine, one half pint of beer, one quarter pint of strong lager.

A heavy drinker is defined as a person who drinks almost every day and becomes intoxicated several times a month. A moderate drinker is defined as a person whose drink does not exceed pint 0.8 gm/Kg. of body weight of ethanol/day upto a limit of 80 gms. or an average of 0.7 gm/Kg. for 3 successive days.

ALCOHOL DEPENDENCE

The following is the DSM-IV diagnostic criteria for alcohol dependence.

A) ATLEAST THREE OF THE FOLLOWING :-

1. Alcohol is often taken in larger amounts or over a longer period than the person intended.
2. Persistant desire or one or more unsuccessful efforts to cut down or control use.
3. A great deal of time spent in activities necessary to get the substance, taking the substance or recovering from its effects.
4. Frequent intoxication or withdrawal symptoms when expected to fulfill major role obligations at work, school or home.

5. Important social, occupational or recreational activities given up or reduced because of substance use.
6. Continued substance use despite knowledge of having a persistant or recurrent social, psychological or physical problem that is caused or exacerbated by the use of the substance.
7. Marked tolerance : need for markedly increased amounts of the substance(atleast 50% increase) in order to achieve intoxication or decreased effect or markedly diminished effect with controlled use of the same amount.
8. Characterstic withdrawal symptoms.
9. Substance often taken to relieve or avoid withdrawal symptoms.

B) SOME SYMPTOMS OF THE DISTURBENCE HAVE HAVE PERISHED FOR ATLEAST ONE MONTH OR HAVE OCCURED REPEATEDLY OVER A LONGER PERIOD OF TIME.

EPIDEMOLOGY:-
1) 14% Life time prevalence for dependence (30% in an Industry).
2) More prevalent in men than women(3:1)
3) Many men and women over age 50 choose abstinence.
4) Half of the alcohol is consumed by 10% of alcoholic drinkers.

5) In HAL Hospital, Bangalore, 35% of the total admission to male wards are due to alcohol drinking.

ETIOLOGY:-

1) Alcohol dependence runs in families

2) Children of alcoholic parents are at high risk of developing alcohol abuse whether or not they are raised by their biological parents

3) The familial association is strong for the male child of an alcohol dependent father.

4) There are ethnic and cultural differences in susceptibility to alcohol and its effects. Asians show sensitivity which is due to structural mutation in the gene that codes for an enzyme involved in alcohol metabolism(aldehyde dehydrogenese). This enzyme is thus unable to metabolise acetaldehyde, a toxic product of alcohol metabolism. such persons show acute toxic effects such as intoxication, flushing, dizziness and headache after consuming only minimal amount of alcohol. This is called "alcohol hot flush syndrome". such cultural groups such as jews,conservative protestents and Asians have lower rate of alcohol dependence, whereas others such as native Americans, Eskimos and some groups of Hispanie men show high rate.

TYPES OF ALCOHOLISM

I. PRIMARY ALCOHOLISM:-
Development of alcohol dependence before the onset of any other type of psychiatric illness.

II. SECONDARY ALCOHOLISM:-
Misuse of alcohol as a consequence of anxiety or some other type of psychiatric illness or onset of alcoholism after emergence of signs & symptoms of another psychiatric illness.

III. ALCOHOLISM,TYPE - I

IV. ALCOHOLISM, TYPE-II
It is important to differentiate between type-i and type ii alcoholism because the causes, therpeutic approach and prognosis differs with the type of alcoholism.

TYPE - I	TYPE - II
1. More common	less common
2. 75% of male alcoholics & almost all women alcoholics	Affects only men.
3. Late onset(after 25 yrs. of age)	before 25 yrs. of age.
4. Mild in severity	Severe

5. Influenced by environmental factors	Not so
6. Guilt & fear about alcoholism - present	Absent
7. No inheritance	Inherited by sons from fathers but not daughters.
8. Anti social personality behaviour & criminality does not co-exist	Exists
9. High frequency of abstinence	Low frequency of abstinence
10. Depression +	Depression ++
11. Attempted suicide +	Attempted suicide ++++
12. Response to benzodiazepines - good	Response to benzodiazepines - poor

ALCOHOL INTOXICATION

Common alcohol induced organic mental disorder. Alcohol acts as a CNS depressant in a manner similiar to that of anaesthetic agents, though very much slowly. Its initial action is on the reticular formation leading to increased excitability of the cortex at alcohol level of 30% mg%. later there are direct toxic effects on the cortical neurons as well, at alcohol level of 50 mg%.

CLINICAL FEATURES:-1. Inebriation

2. Subjective exhilaration

3. Excitement

4. Feeling of superiority & skill

5. Slowed and superficial thinking

6. Poverty of association

7. Poor attention and concentration

8. Increased distraction

9. Impaired reasoning and judgement

10. Faulty learning and retention

11. Reduced acuity of perception.

12. Impaired muscular control leading to ataxia

13. Delayed reaction time

14. Dysarthria

15. Progressive loss of restraint

16. Loss of self control

17. Odd behaviour

18. Emotions of hilarity, sadness & self pity

19. Increasing drowsiness

20. Coma

The above situations occur in graded manner. But the situation is complex depending upon the rate of rise to a given level, thus a quick rise will produce effect at a lower blood alcohol than a gradual rise. Tolerance within the CNS may be obvious in habituees, who absorb more rapidly yet at the same time become resistent to the early effects. prolonged ingestion can also

lead to metabolic adaptation as is evidenced by increased rate of metabolism during periods of long continued chronic intoxication.

PATHOLOGICAL INTOXICATION

This State has been described as a condition in which the person becomes intoxicated on very small amount of alcohol, as little as 60 ml.

CLINICAL FEATURES: 1) Uncontrolable rage.

2) Excitement

3) Seriously destructive actions - persons/property.

4) Delusions

5) Visual Hallucinations

6) Suicide attempts

7) Tendency to epilepsy or hypoglycemia

are some times evoked

Brain damaged persons are particularly liable to this type of intoxication.

ALCOHOL WITHDRAWAL SYNDROME

This important group of manifestations occur against a background of severe alcohol abuse but make their appearance usually after a period of complete or relative abstinence. It seems therefore that they depend not on the direct toxic effects of alcohol present at that time but rather on a fall in the level of circulating alcohol within the blood. The intensity of the withdrawal symptoms is related to the amount of alcohol that has been taken and the duration of consumption.

ALCOHOLIC TREMOR: This is the commonest withdrawal effect, begins 24 -36 hours after the last drink(Phase - I alcohol withdrawal) and is manifested by increasing anxiety, irritability, feeling of shaky inside. Increased autonomic activity resulting in restlessness, hypervigilence, hyperactivity, increased sweating, Tachycardia, increased systolic blood pressure, poor attention and concentration, Ataxia, dehydration, Insomnia and craving for relief drink which further alcohol will bring.

RUMFITS(PHASE II WITHDRAWAL): Can occur at any time from 12 hours to 14 days after the last drink, usually after a heavy consumption. The Rum fits are usually seen after several years of established drinking. Very occasionally they are seen while the consumption continues, presumbly as a result of transient fall in the blood alcohol level.

The fits are mostly of tonic - clonic variety, occur in bouts of 2-6 at a time and very occasionally status epilpeticus may be

precipitated. If focal fits occur one should exclude head injury. In about 30% of cases fits are followed by D.T. conversly 30% of cases of D.T. and 10% of cases of auditory hallucinations are prceeded by fits.

EEG is abnormal during fits. but returns to normal after recovery.

DELIRIUM TREMENS(D.T.)

This is the most serious of the alcohol withdrawal phenomena with a mortality rate of upto 5%.

PREDICTION: It is difficult to predict who will develop D.T. Infection, head trauma, poor nutrition, rum fits and previous episodes of D.T. are the contributing factors towards D.T. More than one out of every three patients who suffer fits develop subsequent D.T.

Clinical features:- If D.T. is to occur, it generally does so within 24 -72 hours after withdrawal but can appear even after a week of withdrawal. D.T. presents in a dramatic manner and appears to have had an explosive onset. It onsets usually at night. The following clinical features are usually observed

in D.T.

1) Loss of appetite
2) Irritability
3) Tremors, which are usually fast in frequency and becomes more pronounced when a person is under stress. The tremors may involve the tongue to such an extent that the person cannot talk. The lower extremites may tremble so that the person cannot walk. The hands may shake so violently that a drinking glass cannot be held without spilling the contents.
4) Hypervigilence
5) Insomnia, may experience vivid nightmares and wakes in panic.
6) Illusions, a cob web on the roof may be misinterpreted as a scorpion, a rope on the floor may be misinterpreted as a snake.
7) Hallucinations, Usually visual
8) Dehydration, due to restlessness, poor intake, increased sweating and fever.
9) Agitation
10) Autonomic disturbance causing flushing, pallor, weak, rapid pulse, mild pyrexia etc.

11) Delusions, usually persecutory or jealousy,

12) Rum fits

Outcome of D.T. : The disorder is usually shortlived, lasting less than 3 days in the majority of cases. Very rarely recurrent phases may be seen over a longer period of time. D.T. typically terminates in a prolonged sleep after which the person feels fully recovered apart from residual weakness and exhaustion. Rarely, prolonged attack of D.T. may clear to reveal and Amnesic syndrome when Wernicke's Encephalopathy had been present, unnoticed during the acute stage. Mortality of patients with epilepsy and alcoholism is considerably higher than that of other group of patients with epilepsy.

Death when it occurs is due to cardio vascular collapse, infection, hyperthermia or self injury during the phase of intense restlessness. Any infective process and particularly pneumonia markedly increases the mortality.

ETIOLOGY OF D.T. 1) Low serum megnesium

2) Low levels of thiamine & nicotinic Acid (in proportionate to the severity of the symptoms)

3) Acute toxic Hepatitis(in 90% of cases)

4) Head trauma

5) Subdural haematoma

6) Renal Disease

7) Hyponatraemia

8) Hypoglycemia

9) Other drug abuse

MANAGEMENT OF D.T. 1) Simple withdrawal symptoms may be treated as OP

2) Hospitalisation for moderate to severe cases.

3) Fluid replacement, atleast 6 L/day

4) Sedation,chlordiazepoxide, 25-100 mgm tds/qid

5) Thiamine, 100 mgm, 1-3 times/day

6) High potency vitamin preparation (5 & 6 are given as prophylaxis against wernicke's Encephalopathy or nicotinic acid deficiency encephalopathy)

7) Head injury, if present should be managed(skull x-ray/CAT of head if needed).

8) Rule out infection (chest x-ray if needed).

9) Rule out concaminant consumption of other sedatives (especially barbiturate)

10) Treat hypoglycemia with glucose

11) Monitor serum electrolyates,blood urea, CBC

12) Order for LFT, HbsAg and ECG

13) Half hourly TPR chart

14) Decrease stimulation

15) Magnesium sulphate, 1 gm.IM, 6th hrly for 2 days.

16) Phenytoin or Carbamazepine to be given routinely when there is past history of rum fits.

ALCOHOLIC BLACKOUT

Periods of intoxication for which there is complete anterograde amnesia and during which the person appears awake and alert. They can last for days with the intoxicated person performing complex tasks, with no subsequent recollection. Brain damaged persons may be more susceptible to this disorder.

ALCOHOL INDUCED PERSISTING AMNESTIC DISORDER

This disorder manifests as disturbance in recent memory as a result of prolonged use of alcohol. This amnestic disorder is rare in persons under the age of 35 years.

WERNICKE's ENCEPHALOPATHY

Also called alcohol encephalopathy which is due to thiamine deficiency resulting from :

1) Poor nutritional intake
2) Alcohol induced malabsorption in the small intestine.
3) Decreased production of thiamine - dependent enzyme transkatalose.

The resulting malnutrition leads to neurological symptoms consisting of

1) Ataxia,
2) Opthalmoplegia,
3) Nystagmus,
4) Global confusional state and coma.
5) Confabulation,
6) Lethargy,
7) Indifference,
8) Mild delirium,
9) Anxiety,
10) Insomnia or fear of the dark.

PROGNOSIS: This is a medical emgerency. The longer the coma remains untreated, the poorer the prognosis.

MANAGEMENT:

1) Thiamine, 100 mgm/day for 3 days
2) If occular motor signs do not improve within several hours of receiving thiamine, magnesium sulphate is given as per D.T.

KORSAKOFF's PSYCHOSIS

This is a chronic condition usually related to alcohol dependence, wherein alcohol represents the large portion of calorie intake for years. Caused by thiamine deficiency.

CLINICAL FEATURES:
1) Retrograde and anterograde amnesia
2) Confabulation(not required for the diagnosis)
3) Disorientation to time, place/or person
4) Polyneuritis

In its chronic stages, the constitutional state subsides and person again becomes alert but typically apathetic, lacking in spontaneity or self direction

TREATMENT: Thiamine, as above

ALCOHOLIC DEMENTIA

This is an organic syndrome that follows prolonged and heavy alcohol use, seen in about 8-9 % of alcoholics. Diagnosed by persistence of dementia for atleast 3 weeks after drinking ceases and exclusion of all other causes of dementia.

CLINICAL FEATURES:
1) Deterioration in previous intellectual abilities
2) Short and long term memory deficit
3) Impaired abstract thinking
4) Poor judgement
5) Impulse control of sufficient magnitude to interefere significantly with work, usual social activities or relationship with others.

6) No loss of consciousness

7) May be associated with delusions/hallucinations

8) Absence of insight

9) Incontinence, impaired mastication and swallowing during end stage

NEUROPATHOLOGY:

1) Cerebral atrophy(mild or moderate), involving the dorsolateral aspects of frontal lobes with enlargement of frontal horns of lateral ventricles

2) Arachnoid thickening with cell degeneration involving particularly smaller pyramidal cells.

3) Disintigration of nerve fibres with dissolution of the myelin sheaths

BIOCHEMICAL CHANGES IN THE BRAIN:

1) P 3 Component is less well developed

2) Impaired cholinergic function

TREATMENT:

1) Supportive

2) Psychotropic drugs if necessary as sedatives and antipsychotics

ALCOHOL INDUCED PSYCHOSIS

This condition usually requires atleast 10 years of alcohol dependence. Male to Female ratio is 4:1. This disorder is clinically similar to schizophrenia.

Clinical Features: 1) Illusions

2) Hallucinations(visual/auditory).

These hallucinations soon become accusatory and threatening. The individual reacts with fear but is fully oriented and realizes that the voices are hallucinations. However, as the accusation persists, the person develops ideas of persecution. Tinnitus is found in many patients who experience hallucination.

The clinical picture is one in which auditory hallucinations occur in the absence of tremor, disorientation, agitation and increased autonomic activity.

ALCOHOL/SUICIDE

Alcohol amounts for about 1/4 of suicides.

RISK FACTORS: 1) Continued drinking

2) Major depressive episode

3) Suicidal communication

4) Poor social support

5) Serious medical illness

6) Unemployement

7) Living alone

As the number of risk factors increase, so does the likelyhood of suicidal outcome.

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Any of the above four factors identify about 70% of alcoholics who commit suicide, all seven identify 83%. Depression is found in nearly 75% of alcoholics who commit suicide. Whether as a complication of alcoholism or occuring alone, depression is the single most responsible factor in suicide. Treatment of the accompyaning depression reduces the risk of suicide.

ALCOHOL/SLEEP

Alcohol affects sleep onset, duration and architecture, reducing the amount of REM sleep and increasing slow wave sleep. But as blood concentration of alcohol falls, it produces rebound and heightened arousal and increasing REM sleep and also recurrent awakening associated with symptoms of alcohol hot flush syndrome. One of the commonest causes of Insomnia is alcoholism.

ALCOHOLISM - BIOLOGICAL AND BLOOD MARKERS

These are the measurable indicators of chronic alcohol intake. Although most are indicative of alcohol's toxic effects and tend to normalise on abstinence, they offer a useful index of remission and relapse.

The Biological markers include:-

1) Elevation in Hepatic enzymes

2) Increased MCV

3) Presence of Isotransferins

4) Altered noradrenergic function(decreased MHPG, Blunted G.H. response to chlonidine)

5) Increased platelet 5 - HT uptake

6) Decreased platelet monoamine oxidase activity

7) Decreased platelet G-protein sensitivity

8) Increased production of Butanedioles after alcohol intake.

9) Decreased endocrine and physiological response to alcohol.

10) Altered P 300 evoked potential

11) Hyper-responsive heart rate in response to stressor i.e, normalised by alcohol.

12) Mild uric acid elevation

13) Increased FFA

14) M.C.V. of 95-100 gms.

15) Gamma glutamyl transferace level of 30-50+

16) Reduction in the production of all types of blood cells, resulting in macrocytic anemia

17) Decreased WBC production

18) Decreased thymus - derived lymphocytes

19) Decreased production of platelets and clotting factors

PHYSICAL ILLNESSES ASSOCIATED WITH ALCOHOLISM

ALCOHOLIC HYPOGLYCEMIA AND KETOACIDOSIS:

Both these two entites are known since more than 50 years. In presence of significant depletion of hepatic glycogen brought on by starvation, alcohol ingestion, impairs hepatic glucose output by

inhibition of gluconeogenesis. This inhibitory effect develops gradually and accounting for relatively slow fall in serum glucose levels and the late onset of symptoms of hypoglycemia after ingestion of alcohol. Most patients are severe chronic alcoholics with heavy alcohol intake, frequently with an increase intake several days or prior to the episode. Classically the patient begins to develop upper abdominal problems often alcoholic gastritis or pancreatitis leading to severe vomiting and inability to take food or for that matter, alcohol. The upper abdominal pain is due to gastritis, pancreatitis, fatty liver, alcoholic hepatitis or any combination of these. Patients may or may not have symptoms of D.T.

LAB. FINDINGS IN ALCOHOLIC KETOACIDOSIS:-

I) CBC

a) Increased WBC b) Hematocrite - variable

II) BLOOD GAS

a) PH - Moderate acidosis

b) Decreased PCO2

c) PO2 - Normal

III) Ketones

a) B-Hydroxybutyrate is increased (4-5 times)

b) Acetoacetate is increased(1-2 times)

c) B-Hyroxybutyrate/Acetoacetate Ratio- >

IV) Lactate - >

VI) Glucose - variable

VII) Hormones

a) Insulin - <

b) Cortisol- >

c) G H - >

d) Glucogon ->

TREATMENT: 1) IV Glucose in large amounts

2) Supportive therapy

ALCOHOL/LIVER

The prerequisite for the diagnosis, a history of excessive alcohol intake, is often dificult to obtain owing to underestimation of intake or even a patient's denial or unwillingness to divulge drinking habits. The assessment of alcoholic liver disease is further complicated by the welknown correlation between the clinical syndromeand three "classic" Pathologic abnormalities, fatty liver, alcoholic Hepatitis and cirrhosis.

Chronic alcoholics without clinical or laboratory evidence of liver disease have histological damage as evidenced by 85% prevalence of cirrhosis in one study. This indicates that even if alcoholics without clinical liver disease undergo biopsy, the disease process will have already advanced to an irreversible stage in some.

Recent reports indicate that 20% of individuals with suspected alcoholic liver disease have acute or chronic viral hepatitis, cholangitis or other causes of liver damage.

CIRRHOSIS:-

Develops in 8-20% of alcoholics. More than 120 to 180 gms of alcohol per day for more than 15 yrs., duration is a predictive factor for Cirrhosis.

Fatty liver, alcoholic hepatitis, iron overload and hepatic carcinoma are other alcoholic liver diseases.

ALCOHOL/PANCREAS

Alcoholic pancreatitis occurs 5-10 yrs., of heavy drinking before the onset of first attack is seen. After the first attack, persons become susceptible to repeated attacks. Acute pancreatitis may take the form of acute necrotizing, acute edematous, acute relapsing and painless varities.

ALCOHOL/G.I.T.

ESOPHAGUS:- Alcohol causes esophageal motor dysfunction, gastroesophageal reflux and carcinoma.

Esophageal varices may pose problems due to bleeding.

STOMACH: - Alcohol stimulates acid scretion, causing gastritis including patchy mucosal erythemia, erosions, petechiae and exudate.

SMALL INTESTINE:- Diarrhea and intestinal malabsorption and may cause dehydration, pottasium, magnesium and phosphate deficiency.

ALCOHOL/SKELETAL MUSCLE

Alcohol can cause acute and chronic myopathy. This is due to direct toxic effect of alcohol on the muscles,

ANAEMIAS: Acute ethanol ingestion causes reduced erythropoiesis with decreased marrow cellularity and produces vasculization of the red cell precursors in the bone marrow similar to that seen in chloromphenicol toxicity. The vacuoles which appear in both erythroid and sometimes myeloid elements disappear a few days after the cessation of alcohol ingestion. these changes cause aplastic anaemia.

FOLATE DIFFICENCY: As many as 75% of severe alcoholics have been found to have an aneamia related to marrow hypo-function. 40% of these anaemic alcoholics have shown megaloblastic hematopoiesis secondary to folic acid defficiency. however folic acid depletion in the tissues alone, with a normoblastic marrow picture is more prevelant in alcoholics.

SEDEROBLASTIC ANAEMIA: Seen in alcoholics with severe folic acid defficiency. This may also be due to deranged pyridoxine function or decrease in enzyme activity involved in heme synthesis.

HEMOSIDEROSIS: Despite the fact that iron defficiency is common among alcoholics because of repeated bleeding from the g.i.t. and hemostatic defects, a large number of alcoholic have increased tissue iron. hemosiderosis may also

be due to repeated blood transfusions given to treat G.I.T. bleeding. Alcoholism may also cause D.I.C.

ALCOHOL/CNS

The following are the CNS complications that occur in alcoholics apart from what has been described earlier.

Alcoholic Polyneuropathy:-This is one of the common nutritional complications affecting the NS of chronic alcoholics. The symptoms begin distally, the legs are invariablly affected first and often exclusively. The onset is usually insidious with a slowly progressive course. The person typically complains of pain, paresthesias and weakness. The nature of the pain ranges from constant dullache to sharp, transiant, lancinating pain. The paresthesias are often burning in nature and as a rule have "glove and stocking" type of distribution, most pronounced in the toes. Slight tactile stimulation may produce intolerable pain and unpleasant sensation. In severe cases, symmetric, distal motor deficits are seen and in advanced cases, significant atrophy may be encountered. D.T.R., especially achilles reflex are typically

reduced or absent. The pathological process in this disorder is axonopathy.

ALCOHOLIC CEREBELLAR DEGENERATION:

Degeneration of the cerebellar cortex specially of the anterior and superior vermis and adjacent hemispheric folia is often seen in upto 27% of the brains of chronic alcoholics WHO show evidence of cerebellar degeneration at atopsy. Clinically this is characterized by wide based gait, truncal atxia, co-ordination defects, nystagmus, dysarthria and tremor.

CENTRAL PONTINE MYELINOSIS:

This is a rare demylinating disease of unknown etiology which has been encountered primarily in chronic alcoholics.

Clinical features include progressive quardriperesis, pseudobulbar palsy(difficulty with speech and swallowing) and partial or complete paralysis of horizontal eye movements. This disorder may lead to coma and death.

STROKE:

Excessive alcohol consumption is believed to increase the risk of stroke by three fold, especially in young alcoholics. Rebound thrombocytosis, altered autoregulation of the cerebral blood flow, cardiac arrhythmias or

hypotension and hyperlipidemia associated with excessive alcohol consumption or its withdrawal have been implicated in the pathogenesis of stroke in alcoholics.

ACCIDENTAL HYPOTHERMIA: Here the level of consciousness declines progressively with decreased body temperature, miotic pupils, decreased pupillary reaction to light, decreased DTR, increased muscle tone are seen. This disorder carries the mortality rate of 30-80%.

FETAL ALCOHOL SYNDROME

Excessive alcohol consumption in pregnancy produces several developmental abnormalities in the fetus. Features include characterstic face (short palpabral fissures, hypoplastic upperlip), mental retardation, growth defficiency. Cardiac and skeletal defects may also be present.

The neonates born to alcoholic mothers exhibit withdrawal symptoms 24-48 Hrs. of birth in the form of sleep disturbances, tremors and feeding difficulties.

ALCOHOL/ENDOCRINE SYSTEM

HPG AXIS:

1) Gynaecomastia
2) Testicular atrophy
3) Impotence
4) Loss of libido
5) Decreased hair growth

6) Decreased prostatic size

7) Oligospermia

HPA AXIS(PSEUDOCUSHING's SYNDROME):

This alcohol related syndrome clearly differs from true cushing's disease in being transient. With abstinence endocrine abnormalities usually resolve over 2-4 weeks. It resumes with alcohol abuse.

Clinical features: 1) Weakness

2) Fatigue

3) Low backache

4) Plethora

5) Moon face

6) Truncal obesity

7) Easy bruisability

ALCOHOL/CANCER

Alcohol itself is not a Carcinogen but may act as a tumor promotor. Squamous cell carcinoma of the head and neck and esophagus are the most important alcohol related cancers associated with alcoholism. Alcohol predisposes to hepato cellular carcinoma. Role of alcohol in rectal and pancreatic carcinoma is a matter of controversy.

ALCOHOL/INFECTIONS

An alcoholic is predisposed to several varities of infections like pneuomonias, pulmonary and extra pulmonary tuberculosis, spontaneous bacteremia, spontaneous bacterial

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peritonitis, bacterial endocarditis, bacterial meningitis STD, HIV, other Viral and fungal infections.

The increased prevelence of infection in alcoholics is due to the following reasons.

I	Mechanical:	a) Decreased consciousness
		b) Impaired closure of glottis
		c) Diminished respiratory excursia
II	Mononuclear Phagocytosis	a) Impaired pulmonary clearance
		b) Impaired antigen trapping
		c) Impaired antigen processing
III	Humoral Factors	a) Depressed Serum bactericidal activity
		b) Disordered Ig. production
		c) Diminished compliment(C-3)synthesis
IV	Cell mediated Immunity	a) Decreased 'T' Cell mytogenesis
		b) Impaired primary sensitization
V	Polymor - phornucler Leukocytosis	a) Leukopenia
		b) Defective chemotaxis
		c) Defective mobilization
		d) Defective adhenrece

ALCOHOL/ELECTROLYTES

Electrolyte abnormalities are common in chronic alcoholics. Some effects of alcohol are direct others are

as a result of poor nutrition or end organ disease accompanying chronic abuse. Some effects are seen only during acute exposure, some during withdrawal and some only as a result of chronic addiction.

MAGNESIUM: Magnesium depletion occurs in alcoholics. While poor nutritional intake and malabsorption may contribute, it is clear that ethanol causes direct renal leak of magnesium even during the first day of consumption.

CALCTUM: Chronic alcoholism predisposes to acute pancreatitis and these person are prone to severe hypocalcemia. Hypocalcemia in this setting may be severe and unresponsive to exogenously administered vitamin D or parathyroid hormone.

ZINC: Alcohol causes direct increase in the urinary zinc excretion and clearence.

PHOSPHATE: Hypophosphatemia is a clincially significant and common metobolic disturbence seen in alcoholics.

SODIUM & POTASSIUM: Acute alcohol ingestion causes decrease in urinary excretion of sodium, potassium and chloride. Potassium depeletion may also be due to inadequate intake, diarrhea, vomitting or from a combination of this. Hypopotassemia may produce muscle weakness or frank paralysis

in chronic alcoholics. This may also be responsible for some ECG changes in alcoholics and may produce life threatening arrhythmias.

ALCOHOL/HYPERTENSION

Recent epidemiological data firmly establishes an association between hypertension and chronic alcoholism. The magnitude of increase in blood pressure in heavy drinkers is variable, but averages about 5-10 mm.Hg. in both systolic and diastolic BP. The effect is some what greater on systolic BP. The prevelence of BP in the hypertensive range is increased 1.5 - 3 fold in heavy drinkers compared to non drinkers or light drinkers. The prevelence of BP greater than 140 mm.Hg. systolic, 90 mm.Hg. diastolic ranges as high as 40-50% in some series. This association is independent of a number of confounding variables including age, adiposity, smoking, race, use of coffee or educational attainment.

There is increased blood pressure both systolic and diastolic during withdrawal period due to severe autonomic signs and symptoms and decreased GABA nergic activity. As withdrawal symptoms resolve over the first 3-5 days BP decreases.

Cortisol also contributes in hypertension in alcoholics. Cortisol is a potential mediator of alcohol induced hypertension. Plasma cortisol increases after acute alcohol ingestion and a "Pseudocushing's" syndrome including hypertension has been described in chronic alcoholics.

ALCOHOL/CARDIOMYOPATHY

Since 1960 the entity of "alcohol cardiomyopathy" has been recognised. It has been suggested that alcohol is the etiologic factor in atleast 50% of cases of congestive cardiomyopathy. Alcoholic cardiomyopathy typically occurs in men 30-55 of yrs. who have a history of chronic alcohol intake over 10-15 yrs of duration.

PSYCHIATRIC MORBIDITY AMONG THE FAMILY MEMBERS OF ALCOHOLICS

Various mental illnesses have been documented among the spouses and the children of alcoholics. The contributing factors include poor interpersonal relationship among the family members, deteriorated marital harmony, financial difficulties, frequent dismissals from the occupation, carelessness of an alcoholic towards the family welfare, unacceptable family environment. The following are some of the psychiatric morbidity seen among the family members of alcoholics.

1. Anxiety disorder
2. Depression, which may lead to suicide
3. Separation
4. Broken homes
5. Illicit sexual relationship
6. Thefts
7. Anti social personality
8. Alcoholism
9. Criminality
10. Lack of social support

pottasium and phosphate depletion or magensium defficiency.

The following table shows the clinical and laboratory features of acute and chronic alcoholic myopathy.

SIGNS & SYMPTOMS	ACUTE	CHRONIC
EVOLUTION	Muscle pain, Swelling, Weakness Rapid(hrs.-few days)	Atrophy, Weakness Slower(weeks-months)
E.M.G	May be normal	Myopathic denervation Potentials
CREATININE -PHOSPHOKINASE	Increased	Often normal
MYOGLOBINURIA	+	-
MUSCLE BIOPSY	Scattered fibre necrosis, Type I fibre susceptibility	Type II fibre atrophy, Denervation
PATTERN OF DRINKING	Binge	Steady
AFFECTED POPULATION	Male Predominance	Male = Female
ASSOCIATED DISEASE	Alcohol withdrawal, D.T.	Nutritional Deprivation, Peripheral Neuropathy

ALCOHOL/BLOOD

Can be divided into 3 major categories (1) those resulting from the direct effects of alcohol (2) those resulting from secondary nutritional defficiency (3) those caused by hepatic disease.

THERAPIES FOR ALCOHOLISM

All the available measures indicate that the end stage of the present century is a time of escalation and changing pattern of alcohol abuse. Alcohol consumption continues to cause serious problems with the annual cost of alcohol associated illness in U.K. estimated in 1986 at 167 million pounds.

Ample evidence is now accumulating that not only can the alcoholic be motivated for treatment but also that he or she can profit from the treatment.

A MODEL FOR CHANGE

PRECONTEMPLATION STAGE: The alcohol abuser here is unaware of having any problem associated with drinking, although others may recognize it.

CONTEMPLATION STAGE: During this stage some realization that a problem exists is aware to an alcoholic.

DETERMINATION STAGE: In this stage a serious commitment to do something about it is made.

ACTION STAGE: Here an appropriate course of action is taken.

MAINTENANCE STAGE: This stage is one of sustain improvement

The above model for change indicates that the family members or the people belonging to the society are the motivating agents in the

precontemplation stage and an alcoholic and the other above mentioned agents are the motivating agents during the contemplation stage. Once the determination stage is reached the action stage is easily motivated and the goal of abstinence is maintained in maintainance stage.

The above stages are achieved through counselling which is undertaken with the alcoholics and their family members and close associates to educate him in detail about the various physical and psychological ill effects of alcohol. In counselling the "sources" of alcohol abuse is clearly identified and is removed or modified. The interpersonal relationship is strengthened and even the alcohol abuser is made to witness the morbidity and mortality among other alcoholics.

ALCOHOL DETOXIFICATION

The majority of those who are physically dependent on alcohol can be safely detoxicated as Inpatients. However, those addicts who are not willing for admission may be detoxicated as Outpatient.

DISULFIRAN: The use of this chemical in the detoxication of alcohol is known as "chemical aversion therapy". Disulfiran is a long acting drug which has been in use for over 35 years. It blocks the normal metabolism of alcohol including interfering with acetaldehyde breakdown and causes very unpleasant reactions that can discourage further alcohol use. It is rapidly absorbed following oral administration. It can also be implanted subcutenously into the abdominal wall. The more alcohol consumed and higher the dose of Disulfiran, the more severe the adverse reaction.

An alcoholic is admitted to the Ward. Investigations to be performed are TC, DC, FBS, LFT, HbsAg, Blood Urea, Creatinine, Serum Electrolytes, ECG. The patient and his family members are counselled and explained in detail about the procedure and the possible complications of the therapy. The written consent is obtained by the patient and his/her spouse. Disulfiran is started 12 hours after the last drink, 3 tablets of 250 mgms. each on the first day, 2 tablets on the second day and 1 tablet on the 3rd day and Aversion Therapy is administered on the following day. Alcohol, about 50-80 ml. is given to the patient. Within 10 minutes after consumption

vasodilatation of peripheral blood vessels is manifested by a red appearance of the skin, cunjunctivae, by 30-90 mts. person may experience throbbing headache, chest discomfort, palpitations, severe exhaustion, restlessness, fear of death, difficulty in breathing, vertigo, nausea and vomiting, pallor, hypotension, tachycardia. Other symptoms include blurred vision, excessive sweating, thirst, confusion. More severe reactions include cardiovascular collapse and sometimes convulsions.

Hence it is essential to monitor the vital signs of the patient very carefully till he recovers fully from the challenge reaction.

The above reaction may make a person aversion to drink again.

FOLLOWUP: Disulfiran, a long acting drug may be prescribed to an alcoholic on daily basis during followup period. It may even be prescribed 2-3 times per week.

Supervision of disulfiran administration is usually vital as most patients stop taking disulfiran. With proper supervision there is good evidence for the effectiveness of this treatment.

As disulfiran also produces severe reactions if alcohol containing substances are absorbed, the person should avoid any products containing alcohol (eg. after shave lotions, cough syrups, fermented curds/butter milk and tonics).

Rarely disulfiran may produce psychosis.

CHEMICAL AVERSION THERAPY IN HAL HOSPITAL(BC):

In HAL Hospital, Bangalore, 457 alcoholics are de-addicted in the last 6 yrs. The following table shows the details:-

MALES	-	446
FEMALES	-	11
AGE OF THE PERSONS	20-30 YRS	77 (17%)
	31-40 YRS	194 (42%)
	41-50 YRS	160 (35%)
	51-60 YRS.	21 (5%)
FOLLOWUP (REMISSION)	1 YR. -	38%
	2 YRS.-	36%
	3 YRS.-	35%
	4 YRS.-	35%
	5 - 7 YRS.-	33%

RELAPSE PREVENTION

This term is applied to discribe what may be sometimes a deliberate, conscious desire to resume alcohol usage. It is undertaken in the following three settings.

I TO INCREASE AWARENESS OF RISK FACTORS FOR RELAPSE:-

An alcoholic is made to be aware of circumstances which are likely to put him at risk of relapse. Main high risk situations are social pressure, interpersonal conflict and emotional states. The person must be educated to keep away from other alcoholics or alcohol parties, efforts are taken to identify the interpersonal conflicts and to strengthen the interpersonal relationship, emotional stress should be diffused and various alternate ways are identified to help an alcoholic to keep the emotions under his control. Change of pear group is an important aspect here.

II. DEVELOP SKILLS & USE OF OTHER APPROACHES TO PREVENT RELAPSE:

Religious, social activities will help to reduce the risk. One should avoid the stressful situation, if he is unable to cope up or find out the ways to modify the stressful situation in a way with which he can cope up.

II. FACILITATING THE ACHIEVEMENT OF MORE GLOBAL LIFE STYLE CHANGE:-

This involves helping an alcoholic to identify source of stress, alter unhealthy habit patterns, adopt healthy activities, learn more effective time management and adopt a more

balanced life style. One relevent balance is that between recreational or self fulfilling activities (wants) and obligatory duties(shoulds). A snap shot someone's life style and possible source of stress may be provided by a dairy of 1 week's activities, with each activity designated "want" or "should". Important to note and discuss with the client will be the overall numerical balance of "wants" and "shoulds", their pattern Optimally for most people "wants" occur at intervals throughout the day, providing recurrent relief from "shoulds" and the kinds of "wants" as most people will appreciate a balance between solitary and social "wants".

Certain factors such as physical exercise, relaxation practice, meditation and possessing a religious or spiritual aspect in one's life may confer protection against stress across a variety of situations.

All the above 3 will help the client to discover the ways to lead a life more resourcefully.

ALCOHOLICS ANONYMOUS(AA)

This is a self help group formed in 1935 by 2 recovering alcoholics(one a physician) for the purpose of rehabilitating alcoholics. "AA" has expanded into an international movement. Members try to live by achieving self help and mutual support. "AA" has been a major contributor to effect assessment and treatment of primary alcoholism but "AA" Counsellors as a rule have trouble with alcoholism secondary to primary psychiatric illness. Sometimes these counsilors resist needed treatment of primary disorders with proper medication, often leading the person into believing that all medications are addictive and equivalent to alcohol.

The group in "AA" will discuss the causes of alcoholism on individual basis and help each other in discovering the alternate ways to avoid alcoholism. The counsillor as a rule is a moderator of the group discussion and encourage the recovering alcoholics in active discussion and he interrupts only whenever necessary.

"AL -ANON" & " AL-A-TEEN"

These are supportive organisations like "AA" but the members of the group consists of family members of alcoholics in "Al-Anon" & adolscent children in "Al-A-Teen"

PROGNOSIS IN ALCOHOLISM

Various studies indicate that the full remission is seen in about 8-28 percent of cases and about one third will

resort to controlled drinking and the rest will continue to drink.

Those individuals who drop out of treatment program, even if they feel they are doing well and have the problem mastered, are more apt to relapse than those who maintain with the program whether it be in a medical or psychiatric setting or self help group. Many alcoholics will have to make repeated attempts to stop drinking before they ultimately do so. Physicians should have patience, are available and non-judgemental at times of crisis to serve their clients well. It is important for physicians to realise that not all of their patients will recover, but neither should they be discouraged from working with alcoholics because of a false pessimism about prognosis.

(POSSIBLE) BENEFICIAL EFFECTS OF ALCOHOL

Little is mentioned about the possible beneficial effects of alcohol but there is evidence in fact that moderate drinking is associated with increased longevity, heavy drinking and abstainers have lower life expectancies than moderator drinkers. This may be due to the increased fibrinolysis activity and also increased HDL which in turn associated with decreased coronory artery disease, but HDL consist of several components and not all are associated with decreased risk of coronory artery disease. An alcoholic is one who does not know when to stop once he starts drinking. However, the ill effects of alcohol over

weighs the beneficial effects. Hence prescription of alcohol for this purpose is not recommended.

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